Electric Blanket Use and Breast Cancer

To the Editor:

Those involved in the Electromagnetic Fields and Breast Cancer on Long Island Study (EBCLIS)[1,2] have reported significant and conclusive findings in the long-running debate of an EMF–breast cancer relationship. Because the EBCLIS used residential EMF measures and considered characteristics related to proposed mechanisms of causation (eg, stratifying by menopause status and use of hormone receptor status), this study has a degree of precision compared with similar studies, which have produced largely inconsistent results. Kabat and colleagues[1] have investigated a broad range of measures of electromagnetic field exposures and electric blanket use, and the thoroughness of the study unexpectedly gives rise to a number of questions about the informativeness of some the variables studied. EBCLIS has provided evidence that risk of breast cancer resulting from EMF will not be revealed using the variables and measures reported. A fundamentally important EBCLIS outcome and an apparent measure of EMF from electric blankets have been left out of consideration.

EBCLIS authors reported no differences between cases and controls in all residential EMF exposure measurements. Although these variables were appropriately not included in the authors' regression model, given the lack of difference in residential EMF fields between cases and controls, the finding of no association between electric blanket use and breast cancer risk could not be surprising.[1] Any effect of EMF fields from electric blankets would need to be effectively larger than that of residential EMF levels; otherwise, background EMF levels might mask the effects of electric blankets. Another possibility is that the effect of electric blanket EMF was buried in the proxy, self-reported measures used to estimate the magnitude of EMF effect and could well have been uncovered if direct measures of EMF strength from electric blankets were taken. Any similarities or differences between cases and controls would be realized immediately by taking quantitative measures of EMF directly from study subjects' blankets. Because EMF is central to the EBCLIS author's hypothesis, these measures could have provided knowledge of the possible success of finding an association. To this end, the authors overlooked an important measure of electric blanket use despite their comprehensive number of variables.

In the study of electric blankets,[1] the authors included a novel variable to test, ie, whether the study subject's windows were opened or closed during the night. They state, and truly so, that this should increase electric blanket cycling through the night, because the blanket tries to maintain a set temperature. However, has this possible effect been accurately modeled? Although the authors presented this intriguing possibility, there is no mention of its reality. If evidence of increased risk of breast cancer with this variable were found, would the study then revisit the phenomenon to see if it exists? This effect could have been modeled appropriately using electric blanket measures and could have provided further strength to their model. Thus, direct measures of electric blanket EMF could have also provided insight into the window open/closed variable considered.

This study[1] provides evidence of no risk of breast cancer resulting from EMF or electric blanket use, or at least convincing support of the fact that any association between the two will not be realized with the methods used. Although the study is comprehensive, the authors need to address a number of basic questions regarding possible differences in residential EMF levels between cases and controls, and they should consider the use of direct measures of blanket EMF levels. There is need for more direct and increasingly quantitative measures to tease out any possibly concealed relationship between EMF and breast cancer.

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The authors respond:

We thank Jason Robarge[1] for his letter regarding our article.[2] Although direct measurements of the electric blankets actually used by the participants might be of interest, such measurements do not necessarily provide a valid assessment of EMF exposure and were not feasible in our study for the reasons that follow.

1. Because our study had a case-control study design and ascertained the past use of electric blankets, it is likely that participants used several blankets over their lifetime. Many of the older blankets would have been discarded and not available for measurements.
2. Furthermore, most of the participants who used electric blankets were former users (77%); as such, their blankets would not have been in use during the study period.
3. Even for current users, blanket measurements would not provide an indication of past exposure. Before 1989, there were 3 blanket manufacturers, and exposure levels produced by these earlier or conventional blankets were higher than in later years.[3] Newer blankets were manufactured using the PTC (positive temperature coefficient) design, which allowed for cancellation of fields and much lower levels of magnetic fields emit-

ted.[4] Most participants who used electric blankets purchased their blankets before 1989 (88%). We found no association when stratifying by use of older blankets or newer blankets, although the number of participants who reported using the newer blankets was small.

4. We interviewed participants during all 4 seasons of the year. Valid measurements of the electric blankets as they were actually used (eg, use when the window was open or closed) would be difficult. Our results are consistent with the findings of 8 previous studies of electric blankets and breast cancer.

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To the Editor:

A recent editorial[1] asserts that the study by Kabat and colleagues[2] has done "as well as an epidemiologic design is capable of doing" in assessing the effect of electric blanket use on breast cancer. However, I believe that the study has a number of severe limitations that render it unlikely the "intriguing hypothesis" of a contribution of exposure to electromagnetic fields (EMFs) by electric blanket use to the risk of breast cancer "can now be put to rest," as the editorial concludes. A study considered to be "negative" has to show at least that 1) the design is compatible with the study hypothesis, 2) the observed effect was below a margin derived from considerations of the mechanism of action, and 3) the power was sufficient to detect an effect at that level. The study lacks all 3 prerequisites.

The authors start from the hypothesis that extremely low-frequency (ELF) EMF could reduce nocturnal melatonin levels, which could in turn lead to elevated estrogen concentrations, thereby increasing breast cancer risk.[3] Although bright light at night suppresses melatonin, there is only equivocal evidence for ELF–EMF to produce such an effect. It has been indicated recently[4] that exposure to ELF magnetic fields increases cell proliferation in the mammary epithelium without affecting pineal or tissue levels of melatonin. Some insight into the action of ELF–EMFs could be gained from the observation[5,6] that ELF–EMFs reduce the inhibitory effect of melatonin on cell growth in breast cancer cells. Hence, the melatonin hypothesis could be reformulated as follows. Exposure inhibits antimitogenic pathways, in particular the action of melatonin, leading to increased turnover rates in either stem cells or precancerous lesions thereby increasing the probability of malignant transformation or reducing latency of manifest disease.

If a promotional effect is assumed, then the design and analysis of this study[2] are inadequate. Because a promoter that acts for a relatively short duration of time only shifts the distribution of age at diagnosis, it either does not affect the overall incidence or only slightly increases it. Clearly, reduction of latency cannot exceed a fraction of exposure duration. Median duration of use in the EBCLIS study was approximately 6 years. Because controls were frequency-matched by age in 5-year strata, any effect must have been greatly attenuated. Even if age at onset were shortened by 50% of exposure duration, incidence by age stratum would remain almost the same. Effects of this type could be studied by cohort approaches applying multistage models of carcinogenicity, as has been done previously for breast cancer.[7]

A further weakness of the article[2] is its lack of any consideration regarding the magnitude of the effect of electric blanket use. If the study hypothesis is true, then we seek an effect size that is compatible with what is known about effects of circulating estrogens and progestins on breast cancer risk. A reanalysis of 51 epidemiologic studies of hormone replacement therapy (HRT) on breast cancer risk[8] reported an average relative risk of 1.35. Recently published hazard ratios from large randomized, controlled trials of HRT[9,10] were 1.26 and 1.27, with a noticeable increase starting approximately 4 years after onset of therapy. Thus, the hypothesized indirect effect of EMFs is not compatible with effects higher than those mirrored in odds ratios of 1.1 to 1.2. Furthermore, an exposure duration of at least 4 years has to be allowed for. Because at present there is evidence for a reversal of the effect of elevated hormone levels, cases with cessation of exposure approximately 5 years before reference date must be considered unexposed. Given these conditions, both the EBCLIS and the Long Island Breast Cancer Study Project have a negligibly small power to detect an effect of electric blanket use.

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The authors respond:

Dr. Kundi[1] raises concerns about the ability of our study[2] to detect an association between the use of electric blankets and breast cancer, on several grounds. The first is that ours was not a cohort study; the second relates to the small magnitude of the expected effect; and the third is that, despite our population-based case-control sample size of 2780 women, power was limited to formally evaluate whether recent electric blanket use or long duration of use increases a woman's risk of breast cancer. In fact, our study was specifically designed to test the hypothesis that ever-use of electric blankets was associated with increased risk of breast cancer, and we had sufficient power to address this question. While cohort studies may be ideal, case-control studies can also provide valuable insight. As to the small magnitude of effect that Kundi projects, individual epidemiologic studies are rarely capable of reliably detecting relative risks on the order of 1.1 to 1.2. We address Kundi's points in light of our results and those of other studies.

We collected detailed information on lifetime use of electric blankets to explore whether various aspects of exposure influence breast cancer development. In contrast to finding an increased risk, the odds ratios for subgroups of ever-users, adjusted for potential confounders, suggest a reduction in both pre- and postmenopausal breast cancer risk, both in current users and in users for more than 10 years. However, the confidence intervals for these estimates cannot rule out a very modest increase in risk. Thus, as with all observational studies, our results need to be interpreted in the context of previous studies on this question. None of the 8 studies we cited previously,[2] including the Nurses' Health cohort study,[3] observed a statistically significant association between electric blanket use and breast cancer. Furthermore, it is striking that none of the studies that looked at duration of use showed any hint of an association, as one would expect for a promoter. Since publication of our paper, a case-control study in blacks[4] reported an increased risk with greater than 10 years of use; however, this result was based on an exceedingly small number of controls with long-term exposure (n = 5). Estimates based on small sample sizes yield unstable results, because the odds ratio could be markedly affected by the misclassification of only a few subjects.

As suggested by Kundi,[1] we examined the mean (± standard deviation) and median years of use in current and former electric blanket users by menopausal status. Current users had considerably more years of use compared with former users (eg, among postmenopausal controls, mean years of use among current users was 19.5 ± 13.4 compared with 6.5 ± 7.0 in former users). There was no difference in years of use between cases and controls stratified by current/former use. We also looked at the mean age at diagnosis of cases by exposure status and menopausal status. The mean age at diagnosis was not younger for current users or former users compared with never users (eg, among postmenopausal cases, mean age was 64.5 ± 7.2 for current users, 63.1 ± 7.3 for former users, and 62.7 ± 8.1 for never users).

Regarding the laboratory evidence, although there are many experiments designed to study the biologic effects of exposure to ELF–EMF, 2 authoritative reviews have concluded that there is no reproducible evidence of such effects.[5,6] Even the 3 papers cited by Kundi seem to be at odds as to whether melatonin is involved in the inhibition of cell proliferation. Finally, of the 3 references Kundi cites in support of a specific mechanism, earlier work by 2 of the lead authors has been discredited.[7,8] Given these considerations, we believe that Kundi's concerns are not well-founded.

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Drinking Water and Cancer

To the Editor:

I very much agree with the commentary[1] of Steenland and Moe, "Epidemiology and Drinking Water, Are We Running Dry?" Their commentary addressed the work of De Roos et al[2] on the relation between nitrates in public drinking water and the risk of cancer of the colon and rectum. Steenland and Moe suggest that, even given a very favorable milieu within which to conduct a study, these motivated and careful investigators were frustrated in finding results that out-stripped the potential sources of error in their investigation.

Why? Steenland and Moe[1] conclude that the study method—retrospective analysis—is not up to this particular task. They first list the dominant characteristics of the types of problems that are addressed in studies involving water-borne contaminants. "Exposures are typically low, fairly homogeneous, and hard to measure retrospectively."

Steenland and Moe[1] press on, pointing out that, from a regulatory perspective, this inability to produce results relatively free from uncertainty is worrisome.

The events of September 11, 2001 have produced many repercussions in our society. One that touches public health and the management of water systems is the specter of willful contamination of water supplies. Much has been written about how to systematically respond to such threats.[4,5] One response has been a surge of innovation in sensor development. Sensors and arrays of sensors are dramatically improving the ability to monitor conditions in the ambient environment for chemical and biologic–agents often in real time and at a fraction of the cost of previous technology. An equal challenge for the risk manager is to effectively translate the increased flow of data from such sensor arrays into a meaningful signal with regards to the existence of a possible threat.

Happily, the epidemiologist is not confronted with the same type of pressures as the manager of a large urban water system. The epidemiologist can benefit from the increased precision of integrating exposure to selected contaminants from this new generation of sensors into longitudinal designs.

Conducting prospective studies that benefit from the existence of the new sensor technology would likely reduce the inherent error and uncertainty in measurements taken in the ambient environment or in operational water distribution systems. This seems clear. Why could not the same technologies be adapted for use in exposure assessment at the level of the participant? The analogy which forms in my mind is the benefit which flowed from the miniaturization of air pollution measurement devices and allowed exposure to be quantified in breathing zones and not on the roofs of public buildings.

Does this mean that the application of new technologies will quell all sources of uncertainty of studying water-borne pollutants? No. The investigator must still rely on judgment in carefully designing the study. Some things remain the same.

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To the Editor:

In the November 2003 issue of EPIDEMIOLOGY, we presented a study[1] on the association between nitrate in public water supplies and the risk of colorectal cancers. An invited commentary on the topic of drinking water studies, written by Drs. Steenland and Moe,[2] noted the difficulties in assessing drinking water exposures retrospectively, and questioned if epidemiologists could be approaching the limit of what we can learn from retrospective designs. We argue that for studies of drinking water contaminants in relation to cancer, retrospective exposure assessment has often been quite adequate, and alternate study designs have limitations that may preclude them from providing a better solution in the near future.

Despite the challenges of retrospective exposure assessment of drink-

ing water contaminants, these studies have contributed importantly to scientific knowledge about cancer, particularly for arsenic and chlorination byproducts.[3] The commentators suggest the prospective study as an improved alternate design because of the possibility of measuring contaminants in individuals' water supplies as well as incorporating intermediate biologic markers which may be relevant for future disease. Studies of arsenic in relation to intermediate biologic effects including micronuclei in urinary bladder cells,[4] and DNA damage in buccal epithelial cells[5] have demonstrated the utility of this approach; however, there are few intermediate effects that have been convincingly associated with the development of cancer.[6] Thus, validation of intermediate markers as cancer precursors must advance considerably before such markers can replace incident cancer as the outcome.

Long-term prospective studies do offer opportunities for more accurate exposure assessment, with the primary advantage being the possibility of sampling the participants' water supplies at the tap, prior to cancer development. This affords a great advantage for participants with private wells, for which few historic monitoring data exist. Nevertheless, the expense of carrying out a long-term prospective study of drinking water contaminants and cancer would necessitate a compelling hypothesis as a rationale for launching the study. Such studies are likely to begin only after substantial evidence accumulates from retrospective epidemiologic studies, supplemented perhaps with a better mechanistic understanding of the carcinogenic process. For these reasons, even when prospective cohort studies have evaluated drinking water contaminants and cancer, they have primarily relied on retrospective exposure data.[7,8]

Substantial opportunity exists for improving retrospective exposure assessment in drinking water studies. Models that incorporate improved information on raw water quality and on how treatment processes affect the formation of disinfection byproducts are currently being applied to earlier case-control studies of cancer.[9] The use of hydraulic simulation models to estimate historic exposure at different points in a water distribution system can also reduce exposure misclassification in retrospective studies.[10,11] The estimation of historic exposures is likely to be further improved when personal factors affecting exposure[12,13] and internal dose[14,15] are used to refine exposure metrics. For example, in our studies of drinking water nitrate and cancer,[1,16–18] we evaluated subgroups of the population who were likely to have increased formation of N-nitroso compounds.

Drinking water is clearly an essential resource for humans, and epidemiologists should continue to conduct studies to determine what constitutes a "healthful" water supply. Improvement of drinking water studies will depend on further validation of intermediate effect biomarkers as predictors of disease, as well as better exposure assessment through greater knowledge of factors affecting variability in the water supply and in individual exposure and response. Especially as improvements in exposure assessment are applied to retrospective studies, they will continue to play an important role in investigating new hypotheses and testing the consistency of observed associations among different populations.

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Kapadia F, Vlahov D, DesJerlais DC, et al. Does bleach disinfection of syringes protect against Hepatitis C infection among young adult injection drug users? *Epidemiology.* 2002;13:738–741.

In Table 1, the percent of cases who never used bleach should be 74%.